In our second case the small omental nodules did not seem a sufficient explanation of the fluid, which further possessed the characteristics of a transudate; specific gravity 1007. In our first case no cause for the ascites save the hepatic growth was discovered. In Litten's case, which was nodular, to be sure, there is no mention of involvement of the peritoneum, yet there was a moderate amount of ascitic fluid in the lower part of the abdomen. In our third case we believe, from the character of the fluid, that the peritoneum itself was the site of multiple metastases. That in sarcoma of the liver other causes for ascites than plugging of the vessels by sarcomatous masses may exist is, of course, true. A portal thrombosis, a sarcomatosis of the peritoneum, an accompanying nephritis or hepatic cirrhosis—as in Musser's case—may give rise to ascites. But the fact should not be overlooked that ascites may occur, and often does occur, in uncomplicated sarcoma of the liver, at least in the secondary infiltrating melanotic form.

We would add a word as to the value of examination of the fluid aspirated from the serous cavities in cases of suspected involvement of the lining membranes in melanosarcomatosis. In Case II. no pigmented cells were found, nor any cells of unusual size or shape or with nuclei showing atypical mitosis. The fluid was, further, of low specific gravity —1007—and resembled therefore in every respect a transudate. In Case III., however, where we believe the peritoneum was involved in the new growth, not only was the fluid of higher specific gravity, 1018, as in the exudates, but the cells were much more numerous, and many of them were large and in all stages of direct division and some revealed karyokinetic figures. Many cells contained reddish-brown pigment granules, as well as fat-drops.

The absence of pain and of gastro-intestinal disturbances and the slight alteration in the blood-findings seemed in Cases II. and III. rather remarkable, when we consider the advanced stage of the disease; for usually great disturbances in appetite and digestive power are met with in advanced malignant growth, and marked deterioration of the blood is commonly found in these conditions.

SUBARACHNOID SEROUS EXUDATION PRODUCTIVE OF PRESSURE SYMPTOMS AFTER HEAD INJURIES.

BY GEORGE L. WALTON, M.D.,
PHYSICIAN TO NEUROLOGICAL DEPARTMENT, MASSACHUSETTS GENERAL HOSPITAL; CLINICAL INSTRUCTOR IN NEUROLOGY, HARVARD UNIVERSITY.

THE practitioner not infrequently meets with cases of head injury in which moderate febrile movement and prolongation of unconsciousness, greater or less in intensity, tend to eliminate simple concussion, while symptoms of irritation, combined with those of depression, unaccompanied by focal signs, point to more or less diffuse cortical lesion. If the discussion were limited to this class of cases it would be perhaps of no vital moment whether the diagnosis of contusion, bruising, or laceration were adopted, provided only the general condition is recognized, and no active steps are taken in treatment. When, however, to this symptom-complex is added a localized paralysis, whether affecting an arm, a leg, or the entire half of the body, and when, in addition, the depth of unconsciousness, for the first day or two at least, is sufficient to cause anxiety, the question of operative interference at once suggests itself. In such cases the importance of most definite ideas regarding the pathology becomes obvious. The autopsy, unfortunately, furnishes comparatively little clue, so that we are forced to rely somewhat upon theoretical considerations, supported as far as may be by actual observation on the operating-table.

It not infrequently happens in these cases that trephining over the area indicated by the paralysis shows negative result beyond revealing a tensely bulging dura, incision of which is followed by free flow, or rather gush, of clear fluid, the brain beneath presenting no laceration or other abnormality beyond, perhaps, œdema.

A few words will be here in place regarding the seat and purpose of the cerebro-spinal fluid. For the purposes of this discussion we may designate the arachnoid as a delicate membrane situated inside the dura and outside the visceral pia. This membrane closely approximates and is hardly separable from the visceral pia over the convolutions, but bridges over the gyri, and at the base of the brain is widely separated from it, leaving a very considerable space, the same conditions obtaining between the cerebellum and the bulb.

Under this membrane in the interval commonly designated the subarachnoid space the greater part of the cortical cerebro-spinal fluid is collected. Increase of cerebro-spinal fluid not only plays a compensatory part in case of lessened blood-pressure and cerebral atrophy, but results also from increased blood-pressure, as in meningitis, and again serves to relieve, according to Tilleau, the intense engorgement following forced expiratory efforts, as, for example, that produced by prolonged coughing. The transudation of this fluid under normal circumstances, as during tranquil respiration and ordinary cardiac systole, is, of course, accommodated for by nature and produces no symptoms. Under abnormal circumstances, however, the bulk of fluid is capable of rapid alteration, as a preservative effort on the part of nature, whether to relieve vascular tension on the one hand or to fill a vacuum on the other.

The transudation of cerebro-spinal fluid is certainly a very rapid process, and no less rapid is the exudation of serum in the cuticle after bruising, whether the fluid shows itself by infiltration of the tissues or by the localized cavity formation known as a vesicle. There is no reason to suppose that the brain is less liable than other tissues to show congestion and œdema after local bruising, or that an exudatory effort on the part of the bloodvessels would be more tardy than elsewhere in a region in which relief of congestion is of vital moment. Whether the accumulation of fluid following a bruise of the brain has simply the characteristics of cerebro-spinal fluid, or whether it partakes more of the nature of serous exudation, is an easy matter to verify by chemical analysis. The opportunity has not presented itself to the writer since commencing this communication, but we may safely assume the process to be exudatory. There is in either event no theoretical reason why rapid local increase of subarachnoid fluid, with or without accompanying œdema of the brain substance, should not follow local bruising and swelling of the cortex, or its general increase follow diffuse injury. We have, in point of fact, something more than theory to rely upon, such accession of fluid being not infrequently demonstrated by operation.

That a more or less diffuse œdema, whether involving the subarachnoid cavity or the brain itself, or both, and whether accompanied or not by marked laceration, may play a part in prolonging the general symptoms of concussion, seems a not improbable supposition; it is not, however, of this class of cases that I would speak at length, but rather of those cases in which local paralysis of more or less temporary nature accompanies such general symptoms, the pathology being here, I apprehend, a local accumulation of fluid under the arachnoid resulting from rapid exudation at the point of most marked bruising, whether the result of direct violence or of *contrecoup*.

Park has drawn attention to the close adhesion of the arachnoid to the pia at the vertex, excepting where the former membrane bridges the gyri, and as a caution in cerebral irrigation he mentions a case of Macewen, in which about two ounces of fluid were injected apparently into the subarachnoid space and retained. In this case a pressure paralysis ensued and lasted four days. This striking though inadvertent experiment absolutely demonstrated the fact that fluid may remain practically imprisoned under the arachnoid, the condition exactly approximating that of a blister of the cuticle.

Local cerebral œdema has been already suggested (Edes) as sufficient cause for temporary paralysis in medical cases, and Bullard especially has called attention to the fact that the pressure of such œdema may extend, in hemorrhage, the paralysis beyond the region implicated in the original lesion, but little attention has been paid to such pathological conditions as I have indicated, resulting from localized trauma. Trauma has been mentioned, *e. g.*, by Quincke[1] among other causes for serous meningitis. The few cases cited by the author, however, in which trauma played a part were of a rather different type from these cases to which I would call attention, the symptoms of pressure being more marked, the optic nerve even becoming atrophied in one case, and blindness following. Lumbar puncture in this case showed marked increase of pressure and a very considerable escape of serum with specific gravity 1008. In another case of chronic hydrocephalus, with acute exacerbation and death at the age of thirteen years, the influence of a fall at the age of five years was very doubtful.

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[1] Quincke: Volkmann's Sammlung klin. Vortrage, N. F., 1893, No. 67; also Deutsche Zeitsch. f. Newenheilkunde, November 12, 1896, 9 Band, 3 and 4 Heft.

The extremely suggestive work of this author deals in general with internal rather than external increase of fluid. Still the analogy is striking between the symptoms of Quincke's serous meningitis and those of the cases under discussion. Slight febrile movement at the onset of acute cases, headache, restlessness, and vomiting (not constant), dulled sensorium, with or without delirium, sluggish pupils, and occasional local paresis, characterize both. The variation in intensity of symptoms is similar. The frequency of optic neuritis, however, followed by atrophy, in Quincke's cases, together with the retraction of the head, affords a decided contrast to these cases under discussion, while, on the other hand, the complete paralysis leading to suspicion of hemorrhage does not appear to be noted in the cases of typical serous meningitis. In fact, lack of definite local symptoms constitutes one of the diagnostic features of the latter disease as distinguished, for example, from tumor.

In the following fairly typical example of the class of cases to which I will call attention the symptoms were so far suggestive of hemorrhage as to lead to serious question of operation :

CASE I.—A boy of six years, with a negative history, excepting that he is said to have had meningitis and hydrocephalus at eighteen months, and that he is said not to be particularly forward in school training, was struck by a bicycle at noon. Was dazed but not unconscious. That evening he had fever and vomited. He slept until midnight naturally, was then restless until daylight, and at 9 A.M. vomited again. At 1 o'clock the next day, when seen by Dr. Percy, the attending practitioner, he was drowsy, and during the afternoon became unconscious, paralysis developing on the right side, including the arm, leg, and face. The right pupil was larger than the left, but reacted to light. The knee-jerks were present, and the plantar reflex was more active on the left. Defecation and micturition were involuntary. There was a contusion behind the left ear, pressure upon which caused him to cry out.

At 9 P.M., when seen by Dr. Scudder and myself with Dr. Percy, the temperature was 102°, the pulse 96. He could be roused more easily than at six o'clock, and at times spoke coherently when questioned. The facial paralysis was less marked, and the right arm and leg were moved somewhat.

The case up to this time had appeared very suggestive of middle meningeal hemorrhage, and if his condition had steadily grown worse instead of slightly improving, at this time operation would have been strongly urged. In view of the improvement, and in consideration of the age of the patient, it was decided not to operate at once, but to have him removed to the hospital, prepared for operation, and carefully watched.

On being brought to the hospital at 11.30 P.M., he was still drowsy, but could be roused a little and asked for water. The pupils had become equal and reacted. The pressure-symptoms had already lessened, the right arm and leg being moved, though not so freely as the left. There was still slight facial paralysis on the right. The knee-jerks were now absent. Behind and a little above the left ear was an area of great tenderness, with œdema of the scalp. No depression was found. The patient was put to bed, with heaters, and a special nurse appointed to watch for increasing coma, paralysis, or spasms.

During the night the patient was restless and turned constantly. In the morning the paralysis had lessened still more. He was much brighter, and took liquids. A slight ecchymosis appeared behind the left ear. Some headache was present. Dejections were involuntary. On the second day of his stay in the hospital--that is, the fourth day after the accident—he was restless and fretful during the night, crying out at times with headache. He was given ten grains of bromide of potassium and an ice-bag applied. Was still somewhat drowsy, but inclined to talk at times. The pressure symptoms had disappeared. On the fourth day of his stay at the hospital the child was apparently well, and eating and sleeping well. He was kept under observation ten days longer, during which time an acute inflammatory throat trouble appeared, evidently a coincidence, and he was discharged apparently well two weeks after admission.

Dr. Percy tells me that about three weeks later diffuse swelling of the left side of the face appeared and persisted for a week. The swelling included the lids and extended to the neck and behind the ear. There was no discoloration except in a slight degree under the eye. Whether this symptom bore any relation to the accident I shall not attempt to decide.

Another case of temporary paralysis, occurring in a child, was seen by me with Dr. Boland, of South Boston, who has kindly furnished the following report:

CASE II.—L. D., aged three and one-half years, was thrown to the floor from a child's doorway swing. Her mother was swinging her carefully, when one end of the rope slipped from the suspending hook, and the child was thrown to the floor, striking on her head. The mother was alarmed because the child only moaned and did not cry. I saw the child within an hour and found her dazed. I gave a good prognosis. However, the mother soon noticed that the child could not hold her head up, did not take notice, though she could be roused, and spoke " Mamma " appropriately, also that she vomited. The next day it was noticed that her left arm was useless, and the left occipital region was tender, or at least she flinched when it was pressed, though no ecchymosis or swelling could be seen. The following day Dr. Walton saw her with me. She was so constipated that I had to give her high rectal enemas to secure an operation.

About three days after the injury it was deemed best to send her to the Boston City Hospital, with a view to operating if paralysis persisted. Over-crowding caused a delay of twelve hours, and then it was noticed that she began to use her left hand. From this on her convalescence was rapid, and she soon showed no remains of the accident. She is now a bright, pretty child of five and a half years, and is as bright in school as any of her age.

This patient also narrowly escaped surgical interference, my hesitation in advising operation arising rather from general considerations than from a definite idea of the pathological process.

I am now inclined to believe that the lesion in each of these children was localized subarachnoid effusion, resulting from a bruise, and followed by rapid absorption of the fluid.

For absolute demonstration that such accumulation of fluid can take place we have to depend rather on the operating than the autopsy-table, and the following case, reported in full by Dr. Brooks and myself in the *Boston Medical and Surgical Journal*, presents a sufficiently obvious illustration :

Case III.—A young woman, thrown from her horse, struck her head violently against a rail, and was carried home unconscious. Four hours later was still unconscious, breathing quietly, with a pulse of 100. The pupils were alike, somewhat dilated, and reacted sluggishly. There was partial paralysis of the left side of the face, and complete paralysis of the left arm and leg. The patient had vomited after the accident. There was a hæmatoma in the right mastoid region. The patient was taken to the hospital.

Restlessness supervened, and consciousness partially returned. Respirations were shallow; there was incontinence of urine. The left arm and leg became rigid.

On the second day the rigidity of the left arm and leg became less marked, and limited voluntary movements appeared. No marked improvement having taken place, however, at the end of thirty-six hours, it was decided to trephine.

Operation. A small trephine button was removed about two and a half inches above the external auditory meatus. The dura, which was in close proximity to the skull, was slightly nicked, and a quantity of clear serum escaped from the opening. The dura was tense and bulging, and on its incision about half an ounce of clear serum spurted out. The brain appeared somewhat œdematous and prominent, but otherwise normal.

On the third day the left arm and leg moved somewhat and were less rigid. The patella reflex was still absent. On the fourth day the movements of the left arm and leg exhibited further improvement, and the patella reflexes had returned. There was less difference between the two sides of the face.

On the fifth day the left side was used much better, and there was little, if any, facial paralysis. From this time on the general condition varied from moderate delirium to somnolence, a fair amount of nourishment being taken by the mouth. The patient gradually sank, and on the fifteenth day the temperature, pulse, and respirations rose rapidly and she died on the sixteenth day. The autopsy revealed no gross lesion sufficient to explain the fatal termination. Two hemorrhages about the size of beans were found in the left frontal lobe, and various minute hemorrhages were scattered over the brain.

The mechanism of death in this case is somewhat obscure, but the number and extent of small hemorrhages show that the blow must have been an extremely violent one, and it would almost seem that the brain was unable to recover from the general disintegration thereby produced. For the unilateral paralysis there seems absolutely no plausible explanation, except the pressure of the fluid, and the relief of paralysis was apparently due to relief of this pressure. The fluid would doubtless have been absorbed in any event in a few days, and it is a question whether the operation had any decided effect one way or the other upon the progress of the case. In reporting this case Dr. Brooks and myself stated that it seemed probable that the diagnosis of cerebral œdema would appear more prominently in the future than in the past as a cause of pressure symptoms, though the relation between these cases and those of so-called serous meningitis had not definitely occurred to us.

That this local accumulation of fluid may appear in other than traumatic cases is shown by a case in which Dr. J. Orne Green operated for temporo-sphenoidal abscess of aural origin. Localized convulsions and paresis appeared later in the opposite arm, giving rise to the assumption that infection had extended to the arm centre. Operation was performed at this point by Dr. Homans; an incision of this membrane was followed by a gush of clear fluid. Further exploration of the brain revealed nothing beyond an œdematous condition. In this case it would appear that some obscure vasomotor influence had produced local transudation. Possibly some obscure reflex nervous irritation determines such conditions, as suggested by Quincke in comparing local cerebral œdema with transient peripheral swellings, as of the face. It is not improbable that the temporary paralyses of general paralytics owe their origin sometimes to local serous exudation.

The practical bearing of this discussion as regards traumatic cases is upon the question of surgical interference. In two of the cases here mentioned operation was undertaken, and in the other two it was seriously considered and prepared for. The result of the operation was in both cases negative, the severity of the underlying condition in each being so great as to preclude recovery. In the other two cases absorption of fluid was apparently rapid and recovery perfect.

It is of great importance to have this class of cases in mind before operating for supposed meningeal hemorrhage. The following diagnostic suggestions were offered by Dr. Brooks and myself, based upon our experience:

1. In case of hemorrhage the unconsciousness, as a rule, after once appearing has steadily deepened.

2. The other symptoms show, as a rule, *steady* and *continuous* progression.

3. The insensibility to pain is generally profound in case of hemorrhage after unconsciousness is established.

4. Inequality of pupils is likely, though not certain to be present, in hemorrhage.

The cases under consideration, on the other hand, have run a comparatively atypical course, and pressure over the hæmatoma has generally given rise to signs of discomfort, long after the unconsciousness has been so profound that no response to questions could be elicited. The pupils have generally been alike, though reacting sluggishly to light. Diminution or absence of knee-jerk has been a fairly constant symptom.

With regard to the question of operation, if the atypical course of the case tends to eliminate hemorrhage, I should favor delay, even though localized paralysis be present. This is especially true in children, not only because they appear particularly prone to temporary paralysis after blows upon the head, but also because operation is borne much less well in early life, and there is always the fear lest the surgical operation may precipitate a fatal issue.

CONCLUSIONS.

First. A severe blow on the head may result, either directly or by *contrecoup*, in a local bruising, congestion, and swelling of the brain-tissue, with serous exudation into the subarachnoid space, either with or without œdema of the brain substance.

Second. If this accumulation of fluid occurs over the motor centres it may be imprisoned so as to cause focal pressure symptoms, simulating meningeal hemorrhage.

Third. This accumulation of fluid is not compensatory, but represents an ineffectual effort toward relief of tension, as shown by the swollen condition of the underlying brain substance when exposed by operation. The mechanism is probably analogous to if not identical with that of the so-called serous meningitis of Quincke.

Fourth. The lesion is self-limiting, the resulting paralysis disappearing in the course of a few days.

Fifth. This condition may be mistaken for middle meningeal or middle cerebral hemorrhage. The diagnosis is difficult and sometimes impossible. Factors aiding in the diagnosis are (*a*) an atypical course, (*b*) absence of steadily increasing coma, and (*c*) the appearance of sensitiveness to pain on manipulation of the head, even after the unconsciousness is so great that questions are not answered. The general symptoms (restlessnes, stupor, headache, and moderate febrile movement) may be the same in both conditions.

Sixth. The mere presence of paralysis following a blow upon the head is not necessarily an indication for immediate operation, and in the absence of steadily deepening unconsciousness and of steady progression of other cerebral symptoms, it will be often wise to postpone surgical interference, though generally speaking an exploratory operation is always justified in case of focal paralysis following head injury.

Seventh. This lesion is to be particularly borne in mind in the case of children and young adults, and perhaps in alcoholic patients. In elderly patients the same set of symptoms points more decidedly toward hemorrhage.

THE PATHOLOGY AND MORBID ANATOMY OF HUNTINGTON'S CHOREA, WITH REMARKS ON THE DEVELOPMENT AND TREATMENT OF THE DISEASE.

By Joseph Collins, M.D.,
PROFESSOR OF NERVOUS AND MENTAL DISEASES IN THE NEW YORK POST-GRADUATE MEDICAL SCHOOL; VISITING NEUROLOGIST TO THE CITY HOSPITAL; ATTENDING PHYSICIAN TO ST. MARK'S HOSPITAL.

Introductory Remarks. *Point of view.* In attempts to solve the problems imposed upon the neurologist by the exigencies of his specialty, many difficulties are encountered. Of these none has the secret of its genesis and being more carefully concealed than the hereditary degenerative diseases. The pathogenesis of the acute inflammatory diseases of the nervous system is an open book. The process is essentially the same as that of acute inflammation in any other tissue, individual attributes and variations being conditioned by structural peculiarities. It is in reality the reaction of the tissues to different forms of irritation, principally bacterial. The degenerative diseases, on the other hand, yield the mystery of their being and causation in a discouragingly slow way. This is true especially of the degenerative diseases that are handed down from generation to generation, such as the hereditary ataxias, choreas, and dystrophies. Although we may apprize ourselves fully of the terminal morbid anatomy of such diseases, it is almost as impossible to infer the pathogenesis of them from a study of their morbid conditions alone as it is from a consideration of the symptoms. In other words, the status of the beginning or original lesion cannot always be inferred from a consideration of the morbid conditions found at the time of death, particularly if the disease has existed a great number of years. This statement may sound heretical, but I am convinced that it does not materially overstep the boundaries of truth. No one can spend his days in the laboratory at work on the central nervous system of individuals who have succumbed to degenerative nervous diseases of

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[1] Read before the Neurological Society, November 2, 1897.